Minireview

StAR-related Lipid Transfer (START) Proteins: Mediators of Intracellular Lipid Metabolism*

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The abbreviations used are: START, StAR-related lipid transfer; StAR, steroidogenic acute regulatory protein; PCTP, phosphatidylcholine transfer protein; StarD#, START domain containing #; GPBP, Goodpasture antigen binding protein; THEA, thioesterase adipose-associated; CACH, cytosolic acetyl CoA hydrolase; DLC-1, deleted in liver cancer 1; PH, Pleckstrin homology; RhoGAP, Rho GTPase activating protein; SAM, sterile alpha motif; IMM, inner mitochondrial membrane; OMM, outer mitochondrial membrane; ER, endoplasmic reticulum; NPC, Niemann Pick C.

R. Soccio, R. Adams, and J. Breslow, unpublished data.
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StAR-related lipid transfer (START) domains are ~210 amino acid lipid binding domains implicated in intracellular lipid transport, lipid metabolism, and cell signaling events. The prototype is the steroidogenic acute regulatory protein (StAR), which transfers cholesterol to mitochondria in steroid hormone producing cells (1). START domains are found in an extensive protein family, including START domain only and multi-domain proteins, but lipid ligands have only been identified in a few cases (2). The human and mouse genomes each have 15 genes encoding START domains (Table I), and phylogenetic analysis divides the family into six subfamilies (Fig. 1A-B) (3).

X-ray crystal structures have been solved for the MLN64 START domain (4), StarD4 (5), and phosphatidylcholine transfer protein (PCTP) (6). All three share the same helix-grip fold (7), with α-helices at the N- and C-termini separated by nine β-strands and two shorter α-helices. The curved β-sheet forms a deep pocket with the C-terminal α-helix acting as a lid, resulting in an internal hydrophobic cavity (Fig. 2). The START structure differs from other hydrophobic cavity lipid binding proteins, such as SCP-2 (8), PITP (9), and the FABPs (10). The PCTP structure was reported with a phosphatidylcholine (PC) molecule in the cavity (6), while the MLN64 and StarD4 structures contain cavities large enough (~850Å³) to accommodate a cholesterol ligand (~740Å³) (5). In each structure, lipid entry or egress would require a major conformational change, most likely opening or unfolding of the C-terminal α-helix lid. In fact, this helix of PCTP has been implicated in membrane binding and PC extraction (11).

Furthermore, StAR can form partially unfolded states (12) and loses helical content upon binding a cholesterol analogue (13). Recent modeling of StAR using structure-based thermodynamics showed that an open-lid conformational state can exist at equilibrium, and that cholesterol binding and lid closure would significantly stabilize the complex (14).
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StAR subfamily: StAR and MLN64

StAR - The rate-limiting step in steroidogenesis is StAR-mediated delivery of cholesterol to the P450 side chain cleavage enzyme (P450scc/Cyp11A1), which resides on the matrix side of the inner mitochondrial membrane (IMM) and converts cholesterol to pregnenolone (Fig. 3, pathway 2) (1,15). After stimulation by pituitary trophic hormones, acute steroidogenesis results from phosphorylation of pre-existing StAR and rapid synthesis of new StAR (15). When StAR is mutated in humans with congenital lipoid adrenal hyperplasia (16) or knockout mice (17), there are marked defects in steroidogenesis by adrenal cortex and gonads. Early studies did not detect StAR mRNA in other steroidogenic organs, placenta and brain (18), but StAR expression was recently shown in both tissues (19,20). However, StAR null fetuses produce normal levels of placental progesterone (16), suggesting alternate steroidogenic mechanisms.

In order for P450scc to act, cholesterol must get to the outer mitochondrial membrane (OMM), across the intermembranous space, and to the IMM. StAR is synthesized as a 37kD protein, but the N-terminal presequence directs mitochondrial import before being cleaved in the matrix, leaving a 30kD protein. Despite its final matrix localization, StAR most likely acts at the OMM (21). In transfection assays and studies with isolated mitochondria, StAR lacking the presequence (N-62 StAR) has equivalent activity to full-length StAR (22,23). In studies using StAR fusions to mitochondrial proteins, StAR at the OMM facing the cytosol is fully active, while StAR in the intermembranous space or matrix is inactive (21). StAR could simply drop-off cholesterol, or alter the OMM to facilitate cholesterol desorption to the IMM (15). An alternate view is that StAR must be imported to act, since data supporting an OMM site of action rely on non-physiological isolated mitochondria or transfection of non-steriodogenic cells (24).

If StAR acts at the OMM, then what is the purpose of mitochondrial import? StAR can act as a cholesterol transfer protein in vitro, as N-62 StAR selectively transfers sterols from
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donor liposomes to acceptor mitochondria (25). However, N-62 StAR lacks target specificity, as other acceptors include trypsin-treated mitochondria, endoplasmic reticulum (ER), and phospholipid vesicles (25,26). Therefore, the presequence may direct cholesterol transfer to mitochondria in preference to other organelles (15). In addition, import may rapidly inactivate StAR (21), as StAR undergoes proteolytic degradation in mitochondria (27).

**MLN64** - MLN64 was cloned as a gene amplified in breast, gastric and esophageal cancers (28,29). While MLN64 could play a causative role in tumorigenesis (30,31), amplification likely reflects close genomic proximity (within 36kB) to the oncogene c-Erb-b2 (Her-2/neu) (28), which is invariably co-amplified (31). The N-terminus of MLN64 includes four transmembrane helices, while the C-terminal START domain is 37% identical to StAR (30). Like StAR, the isolated MLN64 START domain binds (4) and transfers (32) cholesterol *in vitro*, and stimulates steroidogenesis when co-transfected with P450scc (31,33).

MLN64 expression is detected in all tissues (31), and it is a candidate for StAR-independent steroidogenesis in placenta. However, the transmembrane domain of MLN64 localizes it to late endosomes with the START domain facing the cytosol (32,34). Given this localization, full-length MLN64 is relatively inactive in steroidogenesis assays, but proteolysis could release the START domain allowing delivery of cholesterol to mitochondria (31). Supporting this notion, antibodies against the MLN64 START domain detect full-length protein and prominent smaller bands in placenta and transfected cells (31).

MLN64 also functions in cholesterol mobilization from endosomes via the Niemann-Pick C (NPC) pathway. In NPC disease LDL-derived cholesterol accumulates in the late endosome/lysosome compartment, and two causative genes have been identified (35). NPC2 is a soluble endosomal cholesterol-binding protein, while NPC1 is an endosomal transmembrane efflux pump that co-localizes with MLN64 (32,34). Endosomal cholesterol could thus move...
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sequentially from luminal NPC2 through NPC1 to MLN64 and finally to a cytosolic acceptor (Fig. 3, pathway 1) (35). MLN64 mutations have not been reported in NPC disease, but overexpression of the MLN64 transmembrane domain with no START domain results in an NPC phenocopy, with cholesterol accumulation in enlarged endosomes (32). A similar phenotype was observed upon overexpression of MENTHO (MLN64 N-terminal domain homologue), an endosomal membrane protein 70% identical to the MLN64 transmembrane domain but without a START domain (36).

StarD4 subfamily: StarD4, StarD5, and StarD6

StarD4 and StarD5 share 30% amino acid identity and are expressed in most tissues with highest levels in liver and kidney (3). In the co-transfection assay for StAR-like activity, both StarD4 and StarD5 can stimulate steroidogenesis by mitochondrial P450scc. Since they are widely expressed, other roles in non-vesicular intracellular cholesterol transport have been proposed (3,37). Like StAR (38,39), StarD4 and StarD5 may also deliver cholesterol to mitochondrial Cyp27, which generates 27-hydroxycholesterol (Fig. 3, pathway 3). In liver, this is the initial step in alternative bile acid synthesis, a process that may be rate-limited by cholesterol delivery to mitochondria (39). In peripheral cells, 27-hydroxycholesterol may function as an agonist for the LXR nuclear receptors (40), which activate reverse cholesterol transport (41), and as a more soluble oxysterol that can leave cells directly.

StarD4 - StarD4 was identified using microarrays, as hepatic StarD4 mRNA is decreased 2-3 fold on a high cholesterol diet (3). StarD4 mRNA levels are also sterol-regulated in cultured cells (3), consistent with transcriptional regulation by sterol regulatory element (SRE) binding proteins (SREBPs) (42). Reporter transfection assays have identified a functional SRE in the
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StarD4 promoter. Since StarD4 is coordinately regulated with SREBP2 target genes involved in cholesterol synthesis, it may transport a cholesterol precursor sterol to facilitate this process.

**StarD5** – StarD5 is not regulated by sterols, but ER stress by various agents activates StarD5 expression 2-4 fold in cultured cells. Cells respond to perturbations in ER function by the unfolded protein response (UPR), which activates transcription of ER stress response genes. The ER is a sterol-poor membrane despite being the site of cholesterol synthesis, so sterol transport by StarD5 may help reduce ER stress.

**StarD6** - StarD6 expression is limited to the testis, in germ cells at all developmental stages, but not in somatic Leydig and Sertoli cells. StarD6 could be important for male fertility, as sterols play key roles in germ cells, from meiosis to capacitation, and high concentrations of desmosterol in sperm tails may facilitate motility.

**PCTP subfamily: PCTP, StarD7, StarD10, and GPBP**

Unlike the other subfamilies, PCTP, StarD7, StarD10, and Goodpasture antigen binding protein (GPBP) do not share a common exonic organization or homology outside the START domain. In the PCTP structure, 28 residues in the hydrophobic cavity contact PC, and 20 of these are identical or similar in StarD7, including 9 of the 11 aromatics. This suggests that StarD7 may also bind PC, while StarD10 and GPBP exhibit much less similarly at these key residues (only 1 and 3 of the 11 aromatics conserved, respectively), suggesting different ligands.

**PCTP** - PCTP is an extremely specific lipid binding protein, as it promotes intermembrane transfer of PC but not other phospholipids or sterols. PCTP is a cytosolic protein, but it relocates to mitochondria upon clofibrate treatment in some cell types, and this movement requires a putative phosphorylation site at serine 110. PCTP is widely expressed with highest levels in liver, and a function has been proposed in the selective secretion of...
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PC into bile (Fig. 3, pathway 5). However, PCTP knockout mice were reported with normal biliary PC levels, but even wild-type adults in this study had very low liver PCTP protein levels compared to newborn pups (50). PCTP may also play a role in cellular lipid efflux via ATP binding cassette A1 (ABCA1), which forms plasma high density lipoproteins (HDL) by efflux of phospholipids and cholesterol to apolipoprotein AI (41). Overexpression of PCTP results in a dose dependent acceleration of this efflux, suggesting that PCTP replenishes the plasma membrane with PCs that have been removed by ABCA1 (Fig. 3, pathway 4) (51).

**StarD7** - There are no publications on StarD7, but over 700 expressed sequence tag cDNA clones (ESTs) from many tissues indicate abundant and widespread expression.

**StarD10** - StarD10 mRNA is detectable by Northern Blot in testis, liver, kidney, and intestine (52), and there are ~700 ESTs from many tissues. In testis StarD10 is expressed in germ but not somatic cells (52), similar to StarD6.

**GPBP** - The widely expressed GPBP has an N-terminal Pleckstrin homology (PH) domain, two serine-rich domains, and a C-terminal START domain (53). GPBP binds and phosphorylates Goodpasture (GP) antigen, the C-terminus of human collagen IV α3 which is the target of autoantibodies in GP syndrome (53). PH domains mediate protein:protein or protein:membrane interactions, and the GPBP PH and first serine-rich domains are sufficient for binding GP antigen (53). GPBP lacks a conventional serine/threonine kinase domain, but activity is absent in a truncated protein missing the C-terminus (53). The location of the kinase active site is not known, but the START domain is more likely regulatory than catalytic. GPBP mRNA is alternatively spliced and the most abundant form (GPBPΔ26) lacks the second serine rich domain (54). Antibodies that recognize the rarer full length protein but not GPBPΔ26 stain endothelial basement membranes - particularly in renal glomeruli and lung alveoli, which are affected in GP syndrome - and show markedly increased staining in skin biopsies from patients
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with dermatologic autoimmune conditions (54). GPBP is implicated in human autoimmune diseases via association with GP antigen, but other roles are likely as GPBP is conserved in mammals without the GP antigen on collagen IV, and even in *Drosophila* and *C. elegans*.

Acyl CoA Thioesterase Subfamily: CACH and THEA

Cytosolic acetyl CoA hydrolase (CACH) and thioesterase adipose-associated (THEA) each have two type II acyl CoA thioesterase domains and a C-terminal START domain. Multiple subcellular compartments contain acyl CoA thioesterases, which hydrolyze acyl-CoAs to free fatty acids. Several are regulated by peroxisome proliferator activated receptors (PPARs) and nutritional factors, yet their precise roles in lipid metabolism remain undefined (55).

**CACH** - CACH has high hydrolase activity for acetyl-CoA (C₂), low activity for short chain acyl-CoAs (C₄-C₆), but no activity for medium (C₁₂) and long (C₁₆) chain acyl-CoAs (56). Rat CACH activity is detected only in liver and kidney cytosol (57), and hepatic activity is regulated in several metabolic states. First, both starvation and a fat-free diet increase activity, suggesting that CACH regulates acetyl CoA levels for fatty acid oxidation and synthesis (57). Second, activity is increased by cholesterol feeding or by cholesterol synthesis inhibitors, both of which decrease cholesterol synthesis and may increase levels of the precursor acetyl CoA (58). Third, activity is elevated in acute streptozotocin-induced diabetes, but insulin injection prevents this elevation (58). Finally, the PPARα agonist clofibrate elevates CACH activity 3-fold (59). The recent cloning of CACH will allow more detailed molecular and functional studies (56).

**THEA** - Though the thioesterase domains of THEA and CACH are ~60% identical, no acetyl-CoA hydrolase activity was found for recombinant THEA (56), which instead hydrolyzed medium and long chain acyl-CoAs in preliminary studies (60). Humans produce two THEA splice variants, which vary in relative abundance among tissues and encode different C-termini.
THEA-1 and THEA-2 could differ in lipid binding since their START domains have different C-terminal α-helix lids. Only the THEA-2 C-terminus resembles CACH, and mice express only THEA-2. In mouse brown adipose tissue, THEA mRNA is induced by cold exposure, suppressed by warmth, and decreased by 2.5-fold in genetically obese (ob/ob) mice compared to lean littermates (60). THEA maps to the Dietary obese 1 (Do1) locus on mouse chromosome 4, syntenic to an obesity locus in the Quebec Family Study on human 1p32 (60). These experiments suggest THEA plays a role in energy metabolism.

**RhoGAP subfamily: DLC-1, StarD8, and StarD13**

Rho family small GTPases signal in actin cytoskeletal organization and other cellular processes, and Rho GTPase activating proteins (RhoGAPs) stimulate GTP hydrolysis to inactivate signaling (61). The human genome encodes over 50 RhoGAPs, three of which have START domains. Deleted in liver cancer 1 (DLC-1), StarD8, and StarD13 are ~50% identical proteins, each with an N-terminal sterile alpha motif (SAM, a protein interaction domain found in many signaling proteins), a RhoGAP domain, and a C-terminal START domain.

**DLC-1** - The widely expressed tumor suppressor DLC-1 is frequently deleted homozygously in primary hepatocellular carcinomas (HCC) and breast tumors (62). Deletion or loss of expression, perhaps due to promoter hypermethylation, is found in many tumor cell lines from liver, breast, colon, and prostate (62). DLC-1 transfection inhibits growth of DLC-1 negative HCC lines (63) and breast carcinoma lines, preventing in vivo tumorigenicity of the latter (62). In vitro studies identified two potential signaling functions for rat DLC-1, activation of phospholipase C-δ1 and RhoGAP activity for RhoA (64). Phospholipase C-δ1 induces Ca\(^{2+}\) release, and cells microinjected with DLC-1 show a rapid rise in intracellular Ca\(^{2+}\) (65). RhoA affects the cytoskeleton, and cells transfected with DLC-1 round and detach from the plate,
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changes blocked by constitutively active RhoA. The SAM and START domains of DLC-1 were dispensable for this cytoskeletal reorganization (65), although they could play a role at physiological expression levels.

StarD8 and StarD13 - There are no publications on StarD8 and StarD13, which could share redundant functions with DLC-1 or be expressed in different tissues.

The StarD9 gene

StarD9 is in sequence databases as a partial 1820 amino acid human coding sequence (66) with a C-terminal START domain but no defined N-terminus. The 5’ exon in the partial cDNA has at least 4 kB of coding sequence, and upstream genomic sequence shows up to 9995 bp of coding sequence in this exon. (An exon with ~10 kB of coding sequence is atypical, but the mouse gene shares this feature.) Although additional 5’ exons are possible, this large exon and ten 3’ exons constitute an ORF of 11,334 bp, encoding a 413 kD protein. In the entire predicted protein, only the START domain exhibits homology to other proteins. EST evidence suggests StarD9 is predominantly expressed in the nervous system.

Evolutionary Perspective

The distribution of START proteins in completely sequenced genomes shows the evolutionary history of this domain (7). The pathogenic bacterium Pseudomonas aeruginosa PA1579 gene is similar to mammalian PCTP, suggesting horizontal acquisition from hosts since there are no START genes in other prokaryotes (5). START domains are also absent in the yeast Saccharomyces cerevisiae, but the slime mold Dictyostelium discoideum has the CheaterA START protein, indicating the domain was present in the common ancestor to multicellular eukaryotes (7). CheaterA also includes WD40 and F-box domains, suggesting a role in protein
ubiquitination, and CheaterA mutants preferentially form spores rather than stalks (67). The plant *Arabidopsis thaliana* has 20 START genes, 16 of which are fused to homeodomains, suggesting that ligand binding to these transcription factors may regulate plant development (2). In animals, mammals have 15 START genes, while the fruit fly *Drosophila melanogaster* has only four, which most closely resemble mammalian MLN64, GPBP, PCTP/StarD7, and a RhoGAP (Fig. 1C). The nematode *C. elegans* has three additional START genes – most similar to StAR, StarD10, and StarD5 – for a total of seven (Fig. 1C). *C. elegans* K02D3.2 (a protein 23% identical to StarD5, but only ~17% to StarD4 or StarD6) was recently identified as a gene affecting fat storage in a genome-wide RNAi screen (68). START genes have also been described in animals without completely sequenced genomes, including a START protein in the silkworm *Bombyx mori* that binds carotenoids (69).

**Conclusion**

By binding lipid ligands, START domains could function in net lipid transfer between subcellular compartments or in lipid regulation of cellular signaling events. START proteins likely play significant roles in lipid metabolism, fertility, atherosclerosis, autoimmune disease, and cancer, making them potential drug targets. There has been significant recent progress in the study of some START proteins, while others remain essentially uncharacterized and demand further investigation.
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REFERENCES

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TABLE I. **Nomenclature and chromosomal locations of 15 mammalian START genes.** The gene names in italics are used in this review. All human START genes except CACH and THEA have been assigned formal names StarD1-Star13, but some common names are widely used. Physical map positions (chromosome, position in megabases) in the mouse and human genomes are based on the Ensembl database, 2 Dec 2002 revision (www.ensembl.org). A human StAR pseudogene was described in ref 18, and a potential mouse StarD6 pseudogene in ref 3.

**FIG. 1.** **Phylogenetic analysis of the START family.** A, the 15 human START domains were aligned using ClustalW, and the resulting phylogenetic tree divides the family into six subfamilies (3). B, the domain organization for each protein or protein subfamily, with START domains in green and other domains in blue (PH, Pleckstrin homology; Ser, serine-rich; SAM, sterile alpha motif; RhoGAP, Rho GTPase activating protein; 4TM, four transmembrane; Pre, mitochondrial presequence; Thio: acyl CoA thioesterase). C, the START proteins from *Drosophila melanogaster* (fly) and *C. elegans* (worm) most closely resemble certain mammalian proteins or protein subfamilies.

**FIG. 2.** **START domain X-ray crystal structure.** Like other START structures, StarD4 (5) has four α-helices (A-D, blue) and nine β-strands (red) that form a U-shaped sheet. The C-terminal αD helix may open or unfold to allow lipid binding in the internal hydrophobic cavity.
FIG. 3. Potential roles of START proteins in intracellular lipid transport pathways.

START proteins are green, other proteins are blue. 1, for LDL-derived cholesterol (C) to leave endosomes (end), it may be passed sequentially from NPC2, through NPC1, to MLN64, and then perhaps to another START protein. 2, in steroidogenic cells, StAR delivers cholesterol to Cyp11 (P450scc) in mitochondria (mit), which initiates steroid hormone synthesis. 3, some START proteins may deliver cholesterol to mitochondrial Cyp27 to generate 27-hydroxycholesterol, which can have three fates: 3a, it is hydrophilic enough to leave cells; 3b, it can enter the nucleus (nuc) and bind LXR to activate transcription of genes involved in reverse cholesterol transport; 3c, in liver, it is the initial substrate for alternative bile acid (BA) synthesis, while classic BA synthesis from cholesterol is initiated by Cyp7 in the endoplasmic reticulum (ER). 4, ABCa1 effluxes cholesterol and phosphatidylcholine (PC) to nascent HDL, and PCTP replenishes the plasma membrane with PC, while another START protein may replenish cholesterol. 5, in liver, specialized apical ABC transporters efflux cholesterol, BA, and PC into the biliary cannaliculus (BC), and START proteins may deliver lipids for this efflux.
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Table 1
**Figure 1**

(A) 6 mammalian subfamilies

- PCTP
- StarD7
- GPBP
- StarD10
- StarD8
- StarD13
- DLC-1
- StarD4
- StarD6
- StarD5
- MLN64
- StAR
- THEA-2
- CACH
- StarD9

(B) Protein Domains

- START
- PH - Ser - Ser - START
- SAM - RhoGAP - START
- 4TM - START
- Pre - START
- Thio - Thio - START
- START

(C) Fly

- none
- CG6565
- CG8480
- CG3522
- none
- none
- none
- none

Worm

- C06H2.2
- F25H2.6
- T28D6.7
- F26F4.4
- F26F4.4
- none
- none
- none
- none
Figure 2
Figure 3
StAR-related lipid transfer (START) proteins: Mediators of intracellular lipid metabolism
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